

GII. All pts who improved ejection fraction at follow-up (26 in GI and 14 in GII) had an early GADO washout in > 40% of the MRI defined myocardium at risk at baseline scan. The authors conclude that GADO enhanced MRI can effectively evaluate pts with Q wave and non-Q wave AMI, identifying pts at higher risk and predicting changes in global ejection fraction. This may give MRI a more important role in the clinical investigation of pts with AMI.

#### 986-14 Phase-Contrast Magnetic Resonance Angiography: A New Approach in the Evaluation of Coronary LAD Artery Graft Patency and Flow Following Treatment of LIMA Hypoperfusion Syndrome With Additional Vein Graft

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Perioperative and early postoperative occlusion of a left internal mammary artery (LIMA) conduit may complicate myocardial revascularization leading to the potentially fatal LIMA hypoperfusion syndrome. Subsequent distal placement of a vein graft to the left anterior descending (LAD) artery may lead to concurrent or retrograde LIMA flow. The purpose of this study was to determine early and late postoperative flow rates in those patients who received LIMA and saphenous vein grafts to the LAD artery for treatment of LIMA hypoperfusion syndrome. Nineteen of twenty-one patients who met this criteria were available for magnetic resonance imaging (MRI). Early (< 6 months) and late (> 12 months) postoperative flow measurements of grafts were performed using conventional and a novel segment k-space phase-contrast MRI angiography technique. Early MRI controls revealed that all conduits had adapted to the coronary flow type with predominant diastolic perfusion. Patency rate at the early and late control was 100%. Concurrent flow, flow reversal, or steal phenomena were absent. Mean flow rates were 49.2 ml/min for the LIMA and 72.6 ml/min for the saphenous vein graft, respectively. Based on the flow data obtained with MRI angiography, the use of an additional saphenous vein graft as treatment of LIMA hypoperfusion syndrome does not lead to LIMA occlusion. Conduit flow adaptation to the diastolic predominance occurs in the first 6 months after operation.

#### 987 Coronary Vascular Physiology in Ischemic Heart Disease

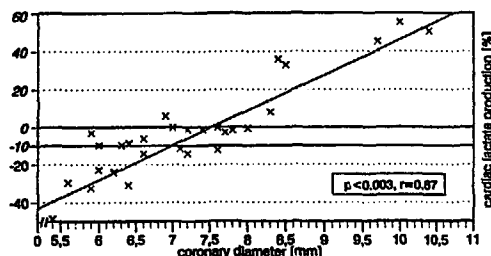
Tuesday, March 26, 1996, 3:00 p.m.–5:00 p.m.  
Orange County Convention Center, Hall E  
Presentation Hour: 4:00 p.m.–5:00 p.m.

#### 987-15 Exercise Induced Myocardial Ischemia in Non-Obstructive Aneurysmatic Coronary Artery Disease Is Related to Coronary Diameters

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Coronary aneurysms (CA) are defined as a dilatation of the coronary lumen  $\geq 1.5$  fold of the normal diameter. CA are often associated with arteriosclerotic stenosis, the potency to cause a myocardial ischemia (CI) as a non-obstructive lesion with an impaired coronary blood flow per se is discussed controversially.

*Aim of the study* was to evaluate CI in patients (pts) with non-obstructive bilateral CA and to quantify its severity depending on the coronary diameters. A coronary sinus lactate study with an incremental atrial pacing (CS) was performed in 31 pts, metabolic criteria for a CI were a reduction of the cardiac lactate extraction < 10% or a frank cardiac lactate production. Coronary angiographies were screened for a segmental to and for movement and deposits of dye known as angiographic stigmata of an impaired coronary circulation.



*Results:* 21/31 pts with CA developed an exercise induced CI in CS with a high correlation of the coronary diameters and the severity of CI ( $p < 0.003$ ,  $r = 0.87$ ) as depicted below for the left anterior descending coronary artery. All pts with a pathological cardiac lactate metabolism furthermore presented with marked angiographic signs of an impaired coronary blood flow ( $p < 0.02$ ).

*Conclusions:* Non-obstructive CA were identified as an entity of ischemic coronary artery disease due to an impaired coronary blood flow. A significant correlation between the coronary diameters and the severity of CI was revealed.

#### 987-16 Intravenous L-Arginine Restores Vascular Reactivity in the Conduit Arteries of Young Hypercholesterolemic Adults

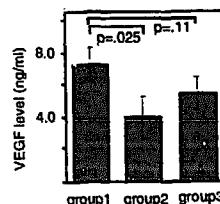
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Hypercholesterolemia (HC) is associated with endothelial dysfunction in the microvasculature of both asymptomatic young adults and in those subjects with clinical evidence of atherosclerosis. This may be reversed by intravenous (IV) L-arginine (L-arg), but the effects on conduit arteries have not been examined. We studied vascular reactivity in 6 HC subjects and 6 controls (LDL-cholesterol  $2.25 \pm 0.62$  mmol/l vs  $4.76 \pm 0.75$  mmol/l,  $p < 0.005$ ), all of whom (age  $29.6 \pm 2.3$  yrs; range 22–36) were asymptomatic, non-smokers, non-hypertensive, non-diabetic and on no cardiovascular medication. Five of the HC subjects had Familial HC and 3 were on cholesterol lowering medication (Simvastatin). Using high resolution ultrasound, we measured brachial artery diameter at rest, in response to reactive hyperemia (endothelium-dependent dilation) and to sublingual glyceryltrinitrate (endothelium-independent dilation). Flow mediated dilation (FMD) was assessed before and after systemic infusion of L-arg (0.1 g/kg body weight) and the effect compared to a 5% dextrose (placebo) infusion. L-arg infusion resulted in a > 20 fold increase in serum arginine levels without change in heart rate or blood pressure in any subject. L-arg produced no change in baseline vessel diameter in either group. However FMD, which was impaired in the HC subjects, improved after systemic administration of L-arg (mean FMD from  $1.1 \pm 0.7\%$  to  $3.9 \pm 0.8\%$ ;  $p < 0.001$ ) compared to controls in whom there was no change (mean FMD from  $3.6 \pm 0.7\%$  to  $5.2 \pm 0.9\%$ ;  $p = \text{NS}$ ). No changes in FMD were observed after the placebo infusion and all subjects had dilation to GTN (HC  $19 \pm 4.6\%$  vs  $18 \pm 1.5\%$  in the controls). Enhanced production or decreased breakdown of NO may mediate the improvement in EDD which occurs in HC but not in normal subjects following IV administration of L-arg. This may be an important anti-atherogenic strategy if it can be achieved by chronic oral administration with L-arg.

#### 987-17 Serum Levels of Vascular Endothelial Growth Factor (VEGF) Are Increased in Patients With Acute Coronary Ischemia

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VEGF, a potent angiogenic protein, stimulates vascular growth in response to ischemia. If VEGF is important in the development of collateral blood vessels, we hypothesized that serum VEGF levels would be elevated in patients with acute ischemic syndromes. To evaluate this, we prospectively collected serum samples and measured VEGF levels in 89 patients undergoing cardiac catheterization. Patients with acute ischemic syndromes (group 1,  $n = 41$ ), significant CAD without symptoms of acute ischemia (group 2,  $n = 22$ ) and no significant CAD (group 3,  $n = 33$ ) were compared. Baseline characteristics were similar between the groups except for a higher incidence of chest pain, heparin use and history of myocardial infarction and a lower incidence of congestive heart failure in group 1. The frequency of collaterals by angiographic core lab analysis was 46%, 71% and 0% in the 3 groups. The serum VEGF levels were 7.3, 3.9 and 5.2 ng/ml respectively.



*Conclusion:* Serum levels of VEGF are significantly higher in CAD patients with acute ischemic syndromes. These findings suggest that VEGF is upregulated in CAD patients with acute ischemia.